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The impact of obesity on seminal fluid in patients with male infertility

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Aim: Data on the effect of obesity on seminal fluid and men fertility are inconsistent. The aim of this study was to evaluate the impact of Body Mass Index (BMI) on semen characteristics.

Method: A cross-sectional study was conducted on 74 infertile men. Semen sample were collected and sperm concentration, progressive motility, total motility and normal sperm morphology were assessed in accordance with WHO 2010 criteria. For each patient weight and height were measure and patients were divided by BMI into normal weight (BMI: 18.5-24.9 kg/m², n=30), overweight (BMI: 25-29.9 kg/m², n=30) and obese (BMI: ≥30 kg/m², n=14). Seminal fluid parameters were compared among the three groups.

Result: Although sperm concentration was lower in obese men, sperm concentration, progressive and total motility and normal sperm morphology did not significantly differ among normal weight, overweight and obese groups (P>0.05).

Conclusion: Our findings suggest that BMI may have no influence on sperm concentration, motility and normal morphology in infertile men.

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The therapeutic effects of matrine for MCD-induced NASH are associated with upregulation of HSP72 and suppression of mTOR

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Non-Alcoholic Steatohepatitis (NASH) is an advanced stage of the metabolic syndrome in liver with serious consequences largely because of hepatic injury, inflammation and fibrosis. Matrine (MW: 248) is used as a prescribed hepatoprotective drug in humans and it has been shown by us to decrease hepatosteatosis and glucose intolerance in high fat-fed mice. Here, we investigated whether matrine exerts therapeutic efficacy for NASH by attenuating hepatic injury, inflammation and fibrosis. The study was performed in Methionine Choline-Deficient (MCD) diet-fed mice for 6 weeks with or without the treatment with matrine (100 mg/kg/d). Compared with untreated MCD-fed mice, matrine markedly reduced hepatic injury (indicated by ALT level, p<0.05), inflammation (indicated by TNF α , CD68 and inflammasome NLRP3, all p<0.05). Along with these effects, matrine inhibited MCD-induced increases in fibrogenesis (as indicated by the expression levels of TGF β , Smad3 and type-I collagen (all p<0.05). Further examination revealed that matrine resecured MCD-suppressed Heat Shock Protein 72 (HSP72, a protective chaperon protein against cell toxicity) and inhibited MCD-activated mTOR (a key master regulator triggering pathogenic pathways leading to NASH). Our findings indicate that matrine attenuated MCD-induced NASH by a new mechanism involving the upregulation of HSP72 and inhibition of mTOR. This hepatoprotective drug may be repurposed for the treatment of NASH.

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